



RECEIVED

SEP 12 2002

TECH CENTER 1600/2900

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Application of: Moore et al.

Application Number: 09/376,430

Filed: August 18, 1999

Title: Cytokine Receptor Common
Gamma Chain Like

Group Art Unit: 1646

Examiner: O Hara, Eileen B.

Attny. Docket No.: PF466P1

#29
HGS
9/16/02DECLARATION OF PAUL A. MOORE UNDER 37 C. F. R. § 1.132Assistant Commissioner for Patents
Washington, D.C. 20231

Sir:

I, Paul A. Moore, do hereby declare and state:

1. I am a citizen of the United Kingdom, residing at 19005 Leatherbark Drive, Germantown, MD 20874.
2. I am a named inventor of the captioned application, which is assigned to Human Genome Sciences, Inc. 9410 Key West Avenue, Rockville, Maryland 20850, (HGS). I have been currently employed, since March of 1993, at HGS where I currently hold the position of Associate Director, Preclinical Discovery at HGS.
3. The work described below was done either by myself or under my supervision or under the supervision of the other named inventors of the captioned application, namely Dr. Steven M. Ruben or Dr. Craig A. Rosen.
4. I have been asked by patent counsel to present evidence of the limited tissue distribution of the CRCGCL receptor protein.
5. The captioned application states that the CRCGCL receptor protein was isolated from an activated T-cell cDNA library. See page 7, line 2. Furthermore, the application describes the limited tissue distribution of the CRCGCL receptor protein in only activated T-cells, as follows:

The tissue distribution in *only activated* T-cells and homology to the cytokine receptors IL2 and IL13 suggests that this protein is a novel member of the cytokine receptor family expressed specifically on T-cells.

See page 10, lines 23-25. (Emphasis added).

6. As further confirmation of the statement quoted in paragraph 5, attached herewith as Exhibit A is a bar graph illustrating the CRCGCL receptor protein's mRNA expression profile in both resting (unactivated) and activated T-cells. RNA was isolated from purified human T-cells that were either untreated or activated. Three modes of T-cell activation were employed: treatment with IL2 and PHA; treatment with anti-CD3 antibody and IL-2; and treatment with IL2, anti-CD3 antibody and anti-CD28 antibody. RNA was subjected to Taqman analysis (a sensitive RNA detection technique) with primers specific for the CRCGCL mRNA and the level of expression standardized to the level of 18S rRNA expression. While no CRCGCL mRNA was detectable in resting T-cells, the CRCGCL mRNA was detectable in the T-cells activated by all three modes of T-cell activation.
7. As the bar graph in Exhibit A demonstrates, CRCGCL receptor protein is upregulated approximately 10-1000 fold only in activated T-cells.
8. I further declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true and further that these statements are made with the knowledge that willful false statements and the like are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of the application or any patents issued thereupon.

6/21/02

Date

Paul A. Moore.

Paul A. Moore, Ph.D.